THE ROCKEFELLER INSTITUTE
FOR MEDICAL RESEARCH
66TH STREET AND YORK AVENUE
NEW YORK 21, N. Y.

November 17, 1952

Dear Josh:

The weather played us a mean trick and I was most disappointed. Had hoped that if you had to be weather bound it would be here as from the airport we were bound for a gathering at the "Sagers" which you would have enjoyed.

The 541, 565 xylose story seems to be as follows: 541 is composed of a mixture of predominantly xyl slows (score as the transducees) a few xyl+ and still fewer xyl-. 565 is in all probability a mutant of the slows and therefore a two gene difference from the wild type + and hence is only transduced to slow by the full plus LT-2. Unfortunately it is here that transduction fails us technically. Study of these intermediates would possibly give us an independent allogenic transformation but the selection is impossible.

Have been mulling over your last letter on phase variation and am disturbed by the following. When LT-2 ^{I} is x- abony ^{II} phase variation remains. If I understood you it should disappear as both the recipient and the donor are in the alternative "released" condition, thus the suppressed antigen in the recipient should be lost with the antigen, paralleling their relationship in FA. The transducee having both antigens in a single cell should not migrate under the conditions of the experiment. Since the transduction does occur and phase variation ensues, the latter would seem to be completely cytoplasmically initiated and controlled, although having a more or less permanent affect on nuclear genes.

Have been doing very little with transduction of late other than checking which of the auxotrophic singles you sent will be stable enough for further analysis. My time is spent grinding up spleens and determining the ratios and total counts of virulent and avirulent cells therein. The results are beginning to come in and I believe we will be able to make some sense out of both the role of heterogeneity in the bacterial population and its relation to nutrition in this disease model.

Bertani's results with phage superinfection seem to clarify and verify my own conclusions with regard to the interference experiments. He found that superinfection with a lytic variant leads to a rapid increase in the amount of this phage present and then its dissappearance, the survivors all carrying the parental phage. More careful analysis of my own variant shows that it is also lytic, at least for certain typhimurium and gallinarum.* The experiment then answered another question for which a more direct answer would have been impossible--Do lytic viruses also transduce?

Received my copy of Stocker's tome today and there is little I can add to your discussion of the publication of the flagellar story to date. I do feel that things should either be not mentioned at all or gone into as far as results permit. Tracks and so forth should be developed, I'm not sure of the phase varaiion story as there apparently are some inconsistencies. Further documentation of phage FA identity should not be included as it is quite irrelevant to the main theme and is the subject of a paper which I was going to discuss with you had we met this weekend.

Sincerely,

Norton

P.S. O. A. has caught up with us at last.

* Pictures enclosed